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The neuropathic foot

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Abstract

The neuropathic foot is described with relation to cause, presentation, dysfunction and identification. The various mechanisms of neuropathic foot lesions are outlined — overload, diabetic gangrene, continuous pressure, direct injury and cutting and temperature effects. The orthotic treatment of the foot is discussed and in particular the importance of proper shoe provision and patient education and indoctrination emphasised. The use of plaster casts and fenestrations to control pressure distribution is described. Finally results of an intensive treatment programme are presented to identify the effect on outcome, as measured by delay in amputation.

Neuropathy

Diabetes is the most common cause of neuropathic feet. Leprosy, syphyilis, pernicious anaemia, lesions to the spinal cord, spina bifida cystica and polyneuropathies of different aetiology cause insensitive feet unprotected because of the loss of pain reaction.

The patient is unaware of this insidious dysfunction. Even when the neuropathy is discovered, there is no mechanism to compensate for this loss of sensory protection. Other ways must be found to avoid lesions of the feet, one of which is education.

Later the patient will notice numbness or tingling in the feet or uncharacteristic pain. Sometimes the pain can be severe, burning or lancinating, with hypersensitivity to touch and sometimes rest pain. The painful neuropathy is self-limited as the sensory loss proceeds.

The sensory neuropathy is often combined with motoric and autonomic neuropathy.

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Dysfunction of the autonomic nervous system gives among other symptoms, anhidrosis with dry, shiny skin, that often is atrophic and easily cracks. The loss of the autonomic sympathetic tone in the peripheral vessels can result in arteriovenous shunting of the blood flow. When the venous blood pressure increases, neuropathic oedema may develop. The thermoregulatory mechanism is also out of function.

Motoric neuropathy mostly affects the short muscles of the foot seen as claw-toes or claw-foot.

Sensory nerve disorder must be diagnosed early to prevent lesions. It is easy to detect, if one just cares to examine the feet. The vibration from a struck 128-cycle tuning fork should be perceived from the toes and at least from the malleoli. For comparison the fork is placed at the skeleton of the hand and in the case where the patient has neuropathic feet, he can evaluate the difference.

Perception of pressure could be tested by a 5.07 Semmes Weinstein nylon probe. Proprioception is examined by letting the patient tell in what direction a toe is moved. If both the Achilles reflexes are missing, there is probably neuropathy.

Mechanism of neuropathic foot lesions

Ulcers and neuropathic bone disorders develop by:

- overload, repetitive mechanical stress and shear;
- diabetic gangrene from metabolic and vascular factors;
- direct injury or cutting;
- continous pressure resulting in ischaemia;
- heat or cold.

Overload

Overload is the primary cause of plantar

ulcers (Brand, 1989). The repetitive compression and friction in the soft tissues under the weight-bearing metatarsal heads generates energy transformed to heat. At temperatures above 44 degrees Celsius the enzymatic system of the cells is ruined and they die. Dependent on the severity of the lesion an inflammatory hyperemic reaction develops and will give:

- callosity of the skin;
- some loss of soft tissue;
- a greater loss of soft tissue, autolysis and a bursa;
- a bigger bursa from continuous damage will cause a breakdown of the skin;
- the repetitive stress might also give a neuropathic osteopathic breakdown or an osteitis from an infected ulcer.

The neuropathic foot is at greater risk from overloading than the normal foot in the same circumstances. The combined sensory and motor neuropathies facilitate the development of ulcers. For example the motor nerve dysfunction makes the short muscles of the foot become paretic, while the long flexors and extensors are active, which causes the common claw-toe and claw-foot deformity. The share of weight-bearing normally taken by the toes is added to the load on the metatarsal heads. Further the sensory loss implies balance dysfunction and makes the patient stand and walk with slightly flexed knees and increases the load on the forefoot (Delbridge *et al.*, 1985; Ctercteko *et al.*, 1982).

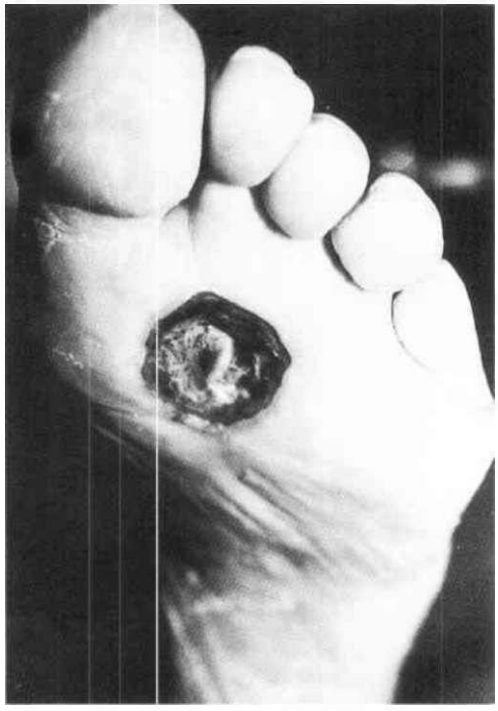

Fig. 1. A neuropathic ulcer of grade 2 exposing the capsule of the metatarsophalangeal joint.

Overload in the presence of normal sensation makes one change to a protective gait, limp or rest, but the neuropathic patient continues to stand and walk. The patient has been walking on the grade 2 deep uninfected ulcer shown in Figure 1 for some months.

Diabetic gangrene

The arteriovenous shunting of the blood by the autonomic nerve dysfunction aggravates any type of oedema. A combination of metabolic disorder, reduced peripheral blood flow and micro-vascular damage may lead to a necrosis of the skin in the central area. It may be the same mechanism for acute osteopathic breakdown.

Continuous pressure

If pressure is applied for a long enough time, it will give ischaemic pressure sores. The lower the foot blood pressure the higher the risk of getting an ulcer.

Direct injury and cutting

Mechanical trauma to the neuropathic foot will occur unnoticed by the patient and lead to complications. The patients must always protect their feet and never walk barefoot or perform any "bathroom surgery".

Heat and cold

With the loss of sense of touch and pain the patient also becomes insensitive to temperature. Further the perception of heat and cold is disordered. The patient may feel his feet are cold although they are not and burn them by exposing them to a heater or water which is too hot. A patient of several years standing forgot his foot in the massage bath while looking at television. He got an ulcer that cost him his foot (Fig. 2).

Orthotic treatment

The majority of patients with neuropathy in the western world have diabetes mellitus.

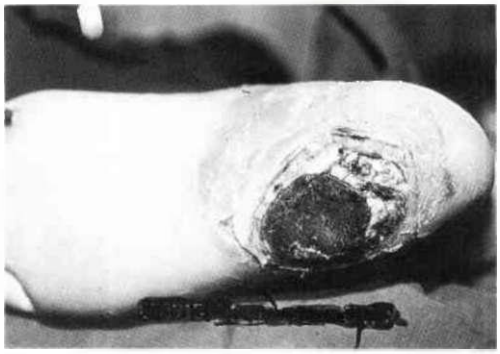

Fig. 2. An infected foot ulcer as a result of an apparatus for massage bath.

Patients with insulin dependent diabetes mellitus will develop neuropathy after ten years' duration of the disease, but with non-insulin dependent diabetes mellitus, the neuropathic symptoms often precede other symptoms of diabetes. Preventive measures are essential to decrease suffering and the enormous economic loss (Malone *et al.*, 1989). Neuropathic lesions of the feet are a defeat to the foot care team.

The orthotic treatment of ulcers is an integrated part of the treatment programme developed by Wagner and built on the Meggit grading of the lesions by severity 0-5 (Wagner, 1981).

Grade 0 designates a foot with no sores, but with a risk of developing an ulcer because of unfavourable loading, deformity, shoe pressure or other factors capable of inducing lesions.

Grade 1 designates a foot with a superficial lesion only involving the skin.

Grade 2 designates a deep lesion extending through the skin down to subcutaneous tissue.

Grade 3 designates a second grade lesion complicated by invasive infection, such as an abscess, arthritis, osteitis or tendon sheath infection.

Grade 4 designates partial gangrene of toes or part of the foot.

Grade 5 designates gangrene of the entire foot, making amputation above the ankle unavoidable.

Apart from meticulous medical care, such as control of metabolism, infection, surgical indications, the feet must be relieved of harmful mechanical factors by orthotic management, which implies:

— education of the patient;
— shoe therapy;
— Unna's paste boots;
— total contact walking casts; and
— other weight-bearing orthoses.

The patients must be educated to follow a well organized life-long treatment programme taught by a team of different specialists to help the patient to co-operate and to promote changes in behaviour.

Shoe therapy

Patients must be motivated to assimilate information and learn about foot care and proper shoes. Every patient is asked to bring all their shoes, old and new, which are examined to find some of the causes of lesions. This is a good way of teaching patients, pointing out which shoes should be avoided and which are proper footwear.

A pair of worn out shoes might be useful by means of cut-outs to unload lesions from pressure (Fig. 3) or used as a temporary footwear dressing following foot surgery.

Shoe principles

Basic mechanical shoe principles considered are:

1. the shoe should be fixed to the foot between the lacing and the heel counter;
2. the sole of the shoe should be stiff from the heel down to the axis of the metatarso-phalangeal joints to harmonize with the movements of the foot;
3. the medial side of the shoe should be straight to avoid medial pressure on the big toe;
4. the shoe should be broad enough to avoid causing ischaemic pressure to the side of the first or fifth metatarsal head;

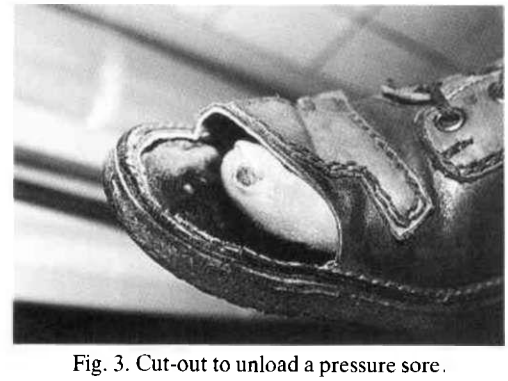

Fig. 3. Cut-out to unload a pressure sore.

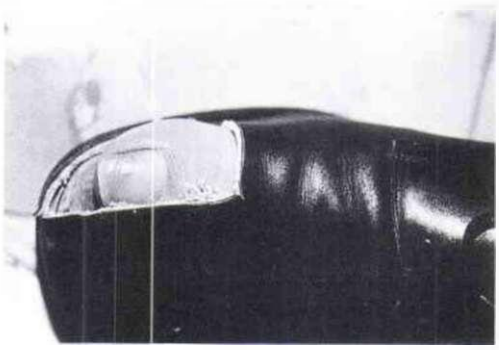

Fig. 4. The narrow toe box of an orthopaedic shoe demonstrated by a cut-out.

5. the toe cap should be spacious enough to give free distance and movement of the toes;
6. the shoe must be 1.5 cm longer than the weight-bearing foot. The tip of the toes must not reach to the toe-cap at toe-off, not even during running.

Very seldom does a patient's shoes fulfil these criteria, which are essential for protection even of a normal foot and Figure 4 shows that even an orthopaedic shoe can have a medial side which is not straight enough for a normal big toe. The patients at risk must be recognized and it must be understood that repetitive stress to the foot and ill-fitting shoes are the most frequent external factors leading to lesions (Apelqvist, 1990; Edmonds *et al.*, 1986; Borssén *et al.*, 1990). As the footwear and walking in combination with loss of sensation are the essential causes of the lesions, it is important to analyse the patients shoes, their mechanical behaviour, examining the soles as well as the inserts to protect each foot from further damage (Bergholdt and Brand, 1975; Mathews, 1988; Holstein *et al.*, 1976).

The feet of grade 0 are subdivided according to Joseph Reed (Coleman, 1988) into five categories, A-E, with respect to protective sensation, healed plantar ulcers, foot deformity and added osteopathy (Table1.)

Principles of relief of weight-bearing

Distribution of the reactive forces from the ground to the foot by inserts is mostly achieved by instinct. The following methods are used to reduce the pressure on an area:

1. elevation of pressure on other areas by pads or build-ups will hopefully reduce the pressure on a selected area, but it is uncertain how the soft tissue is compressed under the weight-bearing parts of the foot skeleton during walking;
2. a mould of the foot in a certain position can, by the hydraulic property of soft tissue, distribute the weight-bearing forces equally to all parts of the foot in that position. During walking however forces are generated inside the foot by the muscle action on the skeleton. The shear forces are unknown;
3. to retain this hydraulic property of the soft tissues of the foot during walking, the foot must be enclosed in a rigid shoe or a plaster cast, hopefully even reducing the propelling action of the foot skeleton;
4. if an area is windowed in a rigid cast or shoe the pressure of weight-bearing is eliminated;
5. when the foot skeleton cannot take weight a rigid ankle tibial condylar weight-bearing orthosis is needed.

Research on foot pressure measurements during weight-bearing and walking are ongoing. Several methods, the EMED system (Schaff, 1987); the pedobarograph (Hughes and Klenerman, 1989; Betts *et al.*, 1980); and the electrodynogram, are used to find ways to measure and control the pressure on the foot sole by design of insoles and shoes.

Table 1. Subdivisions of feet of Grade 0

Category controls	Protective sensation	Healed ulcer	Deformed foot	Shoe measure	Yearly
A	Yes	No	Yes/No	Corrections	1
B	No	No	Yes/No	Load care	2–4
C	No	Yes	No	Footbed+RRBS[1]	3–4
D	No	Yes	Yes	CM-Shoe[2]+RRBS	4–6
E	No	Yes/No	Osteop	CM-Shoe+TCWO[3]	6–9

1: Rigid rocker bottom sole
2: Custom made shoe
3: Tibial Condylar weight bearing orthosis

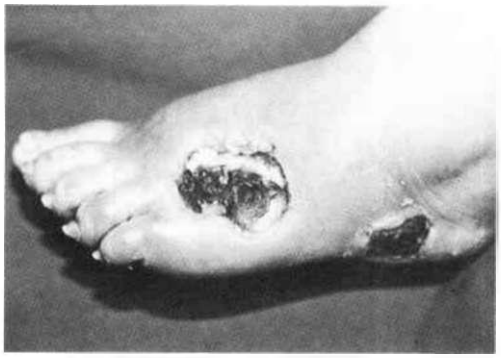

Fig. 5. Central necrosis in a red, hot, swollen and neglected foot, with autonomic neuropathy.

Oedema

Oedema is a precipitating factor to diabetic gangrene and ulcers (Lithner and Törnblom, 1984). The red, hot, swollen foot gives an impression of good arterial perfusion, but on the contrary the nutritive blood flow is reduced by arteriovenous shunting of the blood. A central necrosis of the skin may develop (Fig. 5) and it is urgent to treat the oedema. Besides the treatment of the cause of oedema a zinc paste stocking (Unna's paste boot) should be applied as soon as possible (Fig. 6).

To eliminate the oedema the leg is elevated above the level of the heart during the night and the stocking is applied early next morning.

Hairy legs should be shaved. A tubular stockinette is pulled on. The paste (gelatini zinci oxidi) is turned to solution on a hot water-bath and painted on. Gauze bandage is stuck in small pieces to reinforce the paste. When it is covered, a second layer of paste is brushed on. Generally 3-5 layers are sufficient to give a firm resistance to the muscle contractions so that the venous blood is squeezed centripetally. When the muscles relax, the veins are free to fill which is contrary to the situation with elastic stockings or bandages which always give resilient resistance. The zinc paste stocking is dried by cold air from a hair-drier.

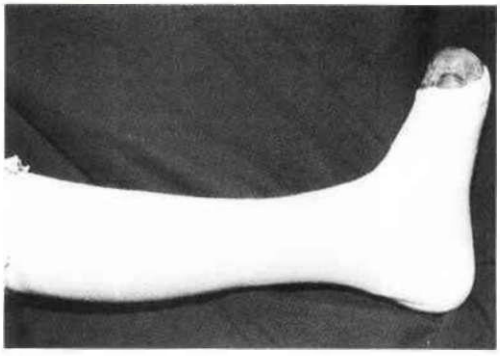

Fig. 6. A zinc paste stocking in the treatment of the oedema. The toes of the foot have been autoamputated.

Total contact casting

Plantar ulcers of grade 1-4 heal more quickly and with less complications in a total contact walking cast (Coleman *et al.*, 1984; Sinacore, 1988; Helm *et al.*, 1984; Mueller *et al.*, 1989; Borssén and Lithner, 1989; Myersen *et al.*, 1992). Ischaemic ulcers of grade 1-2 on the dorsum or sides of the toes could be treated by pressure-relieving cut-outs in a pair of shoes.

Fenestrated walking cast is indicated to;
1. eliminate the pressure on the ulcerated area;
2. immobilize the skin layers and reduce shear;
3. control oedema;
4. maintain ambulation.

Contraindications for walking cast:
1. invasive infection;
2. oedema. Oedema must be minimized before application of the cast;
3. acute osteopathic breakdown. In the case of acute osteopathic breakdown a plaster cast is applied to protect the foot and prevent oedema, but weight-bearing is not allowed.

Cast application

The cast is applied with the patient in a supine position. In a prone position with the knee flexed, the proximal end of the cast will become too spacious, because the heads of gastrocnemius muscle are not stretched during the casting. On a thin stockinette and a minimum of padding on the leg, two rolls of fast setting creamy plaster are applied and carefully rubbed to conform to the shape of the foot and leg till the plaster has set. To make immediate mobilisation and weight-bearing possible the full set cast is reinforced by outer layers of polyurethane impregnated fibreglass. Saw wires make the removal of the synthetic layers easier and when used two tubes should be placed on each side before these layers are applied. The synthetic cast material also provides the support of the sole and the fixation of the heel splint.

A window, just a little bigger than the ulcer is cut out to make it possible to absorb any

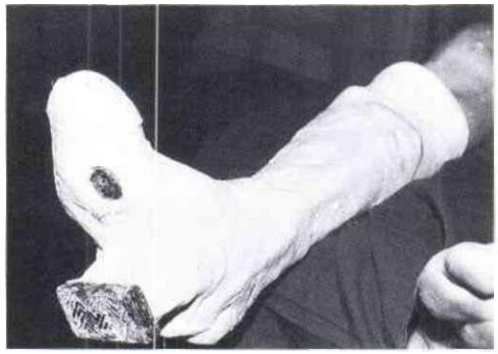

Fig. 7. The walking cast is windowed under the ulcer.

suppuration from the ulcer. The cast in Figure 7 is not reinforced by fibreglass.

The first cast must be checked within a few days. In 10% of the cases the casts become loose and have to be changed because of residual oedema. While changing the walking cast and when obvious healing is taking place, a plaster model is taken from the foot to make a last. Later when the ulcer has healed the individually prepared treatment shoe is ready. It has an individual foot bed made on the last and a rigid rocker bottom sole to give the same mechanical conditions that made the ulcer heal.

In a study of 50 consecutive fenestrated walking casts A. Starkhammar (personal communication) found no fractures, visible deformations or new decubital sores. Six patients developed superficial chafes without effect on the course of treatment.

Casts for heel ulcers are combined with a thermoplastic protector. At 80-90 degrees Celsius the thermoplastic becomes transparent and is fixed with adhesive straps. It can easily be molded to the plaster cast with a wet cold towel. When it becomes cold it will be stiff enough to be taken off without being deformed and further cooled in cold water till it gets hard. The walking cast is fenestrated and the protection replaced, relieving the ulcer from any pressure.

Acute osteopathic breakdown

The diagnosis of neuropathic osteopathy must be made on clinical findings. When roentgenologic signs appear, the breakdown is already in process. The bones must be protected when the inflammatory reaction starts and the foot immobilized in a non-weight-bearing cast. That will arrest the breakdown and give healing with recalcification. Otherwise the architecture of the foot skeleton is lost and so is the weight-bearing function of the forefoot resulting in a requirement for a rocker bottom foot (Fig. 8).

It is difficult to decide how long the patient should be treated without weight-bearing. The neuropathic patient cannot control partial weight-bearing. When increasing weight is taken on the foot, the sign of recurrent breakdown will be a local rise of temperature.

Results

Neuropathic ulcers will mostly heal when relieved from weight-bearing. Healing is dependent on the nutritive blood flow. Ninety-four percent of the diabetic patients with an ankle index >0.7 (ankle blood pressure/arm blood pressure) had ulcers which healed, with an index of 0.45-0.7, 71% healed and <0.45 healing was still achieved in 52% of the patients. Reduced ankle index correlated to a longer time of healing, 47% of 197 diabetic ulcers healed in 3 months, 83% within a year, but still 17% needed more than a year to heal.

The problem in any case is to avoid recurrences and new ulcers. Out of 299 ulcers 194 healed (65%). Eighty-four (43%) remained healed during the time of observation, 3.6-5.6 years, while 66 (34%) healed reulcerations. This makes the education of the patient a very imporant factor.

It is difficult to evaluate the orthotic treatment when there are so many factors involved and to find comparable groups of patients. In a longitudinal study of the diabetics seen at the orthopaedic department of

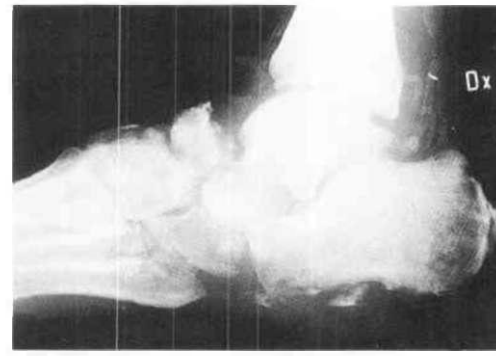

Fig. 8. The second roentgenologic picture of a red, swollen, neuropathic foot left weight-bearing since the first roentgenologic picture was normal.

Norrköping the effect of the total treatment programme was measured by an increase of age at primary amputation and consequently reduced time of survival after primary amputation.

The diabetic amputees' mean age at primary amputation was 72.5 years during the period 1974-1977 and increased by 3.7 years to 76.2 years in 1984-87 (p=0.994). The mean lengths of the amputees' lives was increased 2.6 years (p=0.957) through the same periods. Consequently the mean survival time was 1974-1977, 3.3 years and 1984-87, 2.7 years (p=0.994). In the second group 13 out of 117 patients are still alive, but will give an estimated mean delay of the primary amputation of 0.4 years. The number of amputations was 5% less than expected. The Swede's mean length of life had an increase of 2.2 years through the same years.

Obviously it is hard to prove that it is the effect of the treatment programme, but the effect of the general improved health would be eliminated when the increased length of life is subtracted, but no corrections can be made for a general better care of the diabetics.

Better results ought to be achieved if education and prevention are started early to prevent the normal foot from becoming deformed by traditional shoe fashion.

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